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NEWS 7	Aug	30	IMSworld Pharmaceutical Company Profiles (IMSPROFILES) from IMS HEALTH now on STN
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FILE 'HOME' ENTERED AT 15:22:35 ON 06 OCT 1999

=> **file .bio**

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FULL ESTIMATED COST	1.68	1.68

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FILE 'EMBASE' ENTERED AT 15:27:06 ON 06 OCT 1999

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=> s c-ret or c ret
L1          497 C-RET OR C RET

=> s orphan c-ret
L2            0 ORPHAN C-RET

=> s l1 and (chimer? or fusion)
L3           32 L1 AND (CHIMER? OR FUSION)

=> s l3 and antibod?
L4            1 L3 AND ANTIBOD?

=> dup rem l3
PROCESSING COMPLETED FOR L3
L5           12 DUP REM L3 (20 DUPLICATES REMOVED)

=> d l5 ibib abs 1-12

L5   ANSWER 1 OF 12  Full-text?  CAPLUS  COPYRIGHT 1999 ACS
ACCESSION NUMBER:         1999:325094  CAPLUS
DOCUMENT NUMBER:          131:156079
TITLE:                    Perturbation of RET signaling in the embryonic kidney
AUTHOR(S):                Ehrenfels, Christian W.; Carmillo, Paul J.; Orozco,
                          Olivia; Cate, Richard L.; Sanicola, Michele
CORPORATE SOURCE:         Department of Molecular Genetics, Biogen, Inc.,
                          Cambridge, MA, 02142, USA
SOURCE:                   Dev. Genet. (N. Y.) (1999), 24(3/4), 263-272
                          CODEN: DGNTDW; ISSN: 0192-253X
PUBLISHER:                Wiley-Liss, Inc.
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   We have used a RET-lg fusion protein to disrupt signaling in the
     rat embryonic kidney development pathway. Treatment of embryonic kidney
     organ cultures with RET-lg results in a decrease in branching of the
     ureteric bud and a down regulation in expression of the Wnt-11, Wnt-4, and
     ld genes. These data suggest that Wnt-11, Wnt-4, and ld function
     downstream of RET signaling in normal development. Expression of BMP-7,
     shh, and ptc were uneffected by RET-lg treatment, implying that these
     genes are regulated independently of ret. We have also performed
     immunohistochem. with a GFR.alpha.-1 specific polyclonal antisera to
     localize GFR.alpha.-1 protein expression in the developing kidney.
     c-Ret.

L5   ANSWER 2 OF 12  Full-text?  CAPLUS  COPYRIGHT 1999 ACS
ACCESSION NUMBER:         1998:685118  CAPLUS
DOCUMENT NUMBER:          129:310905
TITLE:                    Study and treatment of diseases related to specific
                          cellular functions of receptor protein tyrosine
                          kinases
INVENTOR(S):              Clary, Douglas
PATENT ASSIGNEE(S):       Sugen, Inc., USA
SOURCE:                   PCT Int. Appl., 81 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ----------------  ----  --------      ---------------  --------
     WO 9845708         A1   19981015      WO 1998-US6842   19980407
         W:  AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
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DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG,
KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX,
NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT,
UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,
FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI,
CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9868876 A1 19981030 AU 1998-68876 19980407
PRIORITY APPLN. INFO.: US 1997-43207 19970408
US 1997-51715 19970703
WO 1998-US6842 19980407

AB The invention relates to methods of evaluating the specific function of a receptor protein tyrosine kinase in cells by activating the receptor in a ligand-independent fashion. In addn., the invention includes methods of identifying compds. that modulate receptor protein tyrosine kinase function. The invention also relates to a method of preventing or treating an abnormal condition caused by an aberration in the function of the **C-RET** receptor, and specifically to the treatment and prevention of neurodegenerative disorders by administering a compd. that modulates the function of the **C-RET** receptor.

L5 ANSWER 3 OF 12 Full-text? MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1999065341 MEDLINE
DOCUMENT NUMBER: 99065341
TITLE: The RET/PTC3 oncogene: metastatic solid-type papillary carcinomas in murine thyroids.
AUTHOR: Powell D J Jr; Russell J; Nibu K; Li G; Rhee E; Liao M; Goldstein M; Keane W M; Santoro M; Fusco A; Rothstein J L
CORPORATE SOURCE: Department of Otolaryngology-HNS Thomas Jefferson University Kimmel Cancer Institute, Jefferson Medical College, Philadelphia, Pennsylvania 19107, USA.
CONTRACT NUMBER: CA21124 (NCI)
SOURCE: CANCER RESEARCH, (1998 Dec 1) 58 (23) 5523-8.
Journal code: CNF. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199903
ENTRY WEEK: 19990301

AB Our research goal is to better understand the mechanisms controlling the initiation and progression of thyroid diseases. One such disease, papillary thyroid carcinoma (PTC), is the leading endocrine malignancy in the United States. Recently, a family of related **fusion** proteins, RET/PTC1-5, has been implicated in the early stages of PTC. Although all five members of this family have the **c-RET** proto-oncogene kinase domain in their COOH terminus, little is known about how these genes alter follicular cell biology. Consequently, to answer questions related to the mechanism of the RET/PTC **fusion** protein action, we have devised a molecular genetic strategy to study PTC using a mouse model of thyroid disease. A new member of this **fusion** oncogene family, RET/PTC3, which has been implicated in more cases of solid tumor carcinoma (79%) than PTC1 or PTC2 and predominates (80%) in radiation-induced thyroid cancer of children, was investigated in our study. We have generated transgenic mice expressing human RET/PTC3 exclusively in the thyroid. These mice develop thyroid hyperplasia, solid tumor variants of papillary carcinoma and metastatic cancer. This new transgenic line will be useful in deciphering the molecular and biological mechanisms that cause PTC and histological variations in humans.

L5 ANSWER 4 OF 12 Full-text? CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1998:746621 CAPLUS
DOCUMENT NUMBER: 130:108431
TITLE: The fetal origin of B-precursor leukemia in the

E.mu.-ret mouse
AUTHOR(S): Zeng, Xiang-Xing; Zhang, Haige; Hardy, Richard R.; Wasserman, Robert
CORPORATE SOURCE: Division of Oncology, The Children's Hospital of Philadelphia, and Department of Pediatrics, The University of Pennsylvania School of Medicine, Philadelphia, PA, 19104, USA
SOURCE: Blood (1998), 92(10), 3529-3536
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: W. B. Saunders Co.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Before the clin. onset of B-precursor lymphoblastic leukemia, E.mu.-ret mice have an expansion of late pro-B cells (CD45R+CD43+CD24+BP-1+) within the bone marrow. To characterize the early effects of the transgene product on lymphopoiesis, the authors initially sequenced the Ig heavy chain (IgH) rearrangements within the late pro-B cells in 24-day-old E.mu.-ret and transgene neg. mice. In both mouse populations, the IgH rearrangements were polyclonal, predominately nonproductive, and exhibited similar V, D, and J gene usage. However, the frequency of N regions, a marker of postnatal lymphopoiesis, was notably different. At the VD junction, N regions were found in 25 of 25 (100.0%) rearrangements from transgene-neg. mice compared with 12 of 36 (33.3%) rearrangements from E.mu.-ret mice. At the DJ junction, N regions were found in 21 of 25 (84.0%) rearrangements from transgene neg. mice compared with 4 of 36 (11.1%) rearrangements from E.mu.-ret mice. Subsequently, the authors sequenced the clonal IgH rearrangements from 9 leukemias that developed in 10-to 38-wk-old mice and found that 7 leukemias had a least 1 rearrangement that lacked N regions at the DJ junction. In addn., V replacement events were obsd. in the 1 leukemia studied in detail. Terminal deoxynucleotidyl transferase, the enzyme responsible for N region addn., was expressed at markedly lower levels in late pro-B cells from 7- to 10-day-old E.mu.-ret mice compared with transgene-neg. mice. Examn. of fetal lymphopoiesis in E.mu.-ret mice identified a relative increase in early (CD45R+CD43+CD24+BP-1-) and late pro-B cells and a decrease in more differentiated CD43- B-lineage cells. Fetal early pro-B cells from E.mu.-ret mice proliferated threefold to fivefold greater but differentiated to a lesser extent than those from transgene neg. mice when cultured in vitro with interleukin-7. These data suggest that the B precursor leukemias in adult E.mu.-ret mice arise from the progeny of pro-B cells generated in utero.

L5 ANSWER 5 OF 12 Full-text? MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998187649 MEDLINE
DOCUMENT NUMBER: 98187649
TITLE: Shc and Enigma are both required for mitogenic signaling by Ret/ptc2.
AUTHOR: Durick K; Gill G N; Taylor S S
CORPORATE SOURCE: Department of Chemistry and Biochemistry, University of California, San Diego, La Jolla 92093-0654, USA.
CONTRACT NUMBER: DK13149 (NIDDK)
T32 CA09523 (NCI)
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (1998 Apr) 18 (4) 2298-308.
Journal code: NGY. ISSN: 0270-7306.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199807
ENTRY WEEK: 19980704

AB Ret/ptc2 is a constitutively active, oncogenic form of the **c-Ret** receptor tyrosine kinase. Like the other papillary thyroid carcinoma forms of Ret, Ret/ptc2 is activated through **fusion** of the Ret tyrosine kinase domain to the dimerization domain of another

protein. Investigation of requirements for Ret/ptc2 mitogenic activity, using coexpression with dominant negative forms of Ras and Raf, indicated that these proteins are required for mitogenic signaling by Ret/ptc2. Because activation of Ras requires recruitment of Grb2 and SOS to the plasma membrane, the subcellular distribution of Ret/ptc2 was investigated, and it was found to localize to the cell periphery. This localization was mediated by association with Enigma via the Ret/ptc2 sequence containing tyrosine 586. Because Shc interacts with MEN2 forms of Ret, and because phosphorylation of Shc results in Grb2 recruitment and subsequent signaling through Ras and Raf, the potential interaction between Ret/ptc2 and Shc was investigated. The PTB domain of Shc also interacted with Ret/ptc2 at tyrosine 586, and this association resulted in tyrosine phosphorylation of Shc. Coexpression of **chimeric** proteins demonstrated that mitogenic signaling from Ret/ptc2 required both recruitment of Shc and subcellular localization by Enigma. Because Shc and Enigma interact with the same site on a Ret/ptc2 monomer, dimerization of Ret/ptc2 allows assembly of molecular complexes that are properly localized via Enigma and transmit mitogenic signals via Shc.

L5 ANSWER 6 OF 12 Full-text? MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97236655 MEDLINE
DOCUMENT NUMBER: 97236655
TITLE: Cell scattering of SK-N-MC neuroepithelioma cells in response to Ret and FGF receptor tyrosine kinase activation is correlated with sustained ERK2 activation.
AUTHOR: van Puijenbroek A A; van Weering D H; van den Brink C E; Bos J L; van der Saag P T; de Laat S W; den Hertog J
CORPORATE SOURCE: Hubrecht Laboratory, Netherlands Institute for Developmental Biology, Utrecht.
SOURCE: ONCOGENE, (1997 Mar 13) 14 (10) 1147-57.
Journal code: ONC. ISSN: 0950-9232.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199706
AB The **c-ret** proto-oncogene encodes a receptor tyrosine kinase which plays an important role in kidney and enteric nervous system development. Germline mutations in **c-ret** are responsible for the dominantly inherited cancer syndromes, multiple endocrine neoplasia types 2A and 2B and familial medullary thyroid carcinoma as well as the developmental disorder Hirschsprung's disease. Using SK-N-MC neuroepithelioma cells stably transfected with an EGFR/Ret **chimeric** receptor, we have studied cellular consequences and signalling events following activation of exogenous EGFR/Ret and endogenous FGF and PDGF receptor tyrosine kinases in cells of neuroectodermal origin. Here we report that Ret activation led to cell scattering, growth inhibition and loss of anchorage-independent growth. Basic FGF, but not PDGF, evoked similar responses in those cells. Nevertheless, activation of all three receptor tyrosine kinases led to ERK2 activation. Analysis of the kinetics of ERK2 activation and downstream events revealed that Ret and FGF receptor activation led to sustained ERK2 activation and SRE transactivation, while PDGF treatment led to transient ERK2 activation and failed to induce SRE transactivation. Our results suggest that sustained, but not transient ERK2 activation may be involved in cell scattering.

L5 ANSWER 7 OF 12 Full-text? MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1998070956 MEDLINE
DOCUMENT NUMBER: 98070956
TITLE: GDNF induces branching and increased cell proliferation in the ureter of the mouse.
AUTHOR: Pepicelli C V; Kispert A; Rowitch D H; McMahon A P
CORPORATE SOURCE: Biolabs, Harvard University, 16 Divinity Avenue, Cambridge,

Massachusetts 02138, USA.
SOURCE: DEVELOPMENTAL BIOLOGY, (1997 Dec 1) 192 (1) 193-8.
Journal code: E7T. ISSN: 0012-1606.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199803
ENTRY WEEK: 19980304
AB The secreted signaling molecule GDNF is expressed in the metanephric mesenchyme and has recently been implicated as a factor necessary for development of the metanephric kidney. We have examined the effects of GDNF on mouse kidney explants. We show that GDNF increases cell proliferation in ureter tips. There is an increase in the number of ureter tips and expansion and **fusion** of adjacent tips and some tips appear to grow toward the source of GDNF. These events are accompanied by transcriptional upregulation of several genes localized to the tips, including its own receptor, **c-ret**, the transcription factor Sox9, and the signal Wnt-11. These results support a model in which GDNF supplied by the mesenchyme regulates growth and branching in the metanephric kidney through the local regulation of ureter tip-specific factors. Copyright 1997 Academic Press.

L5 ANSWER 8 OF 12 Full-text? MEDLINE DUPLICATE 5
ACCESSION NUMBER: 96177666 MEDLINE
DOCUMENT NUMBER: 96177666
TITLE: Breakpoint characterization of the ret/PTC oncogene in human papillary thyroid carcinoma.
AUTHOR: Smanik P A; Furminger T L; Mazzaferri E L; Jhiang S M
CORPORATE SOURCE: Department of Physiology and Internal Medicine, Ohio State University, Columbus 43210, USA.
CONTRACT NUMBER: R29 CA60074 (NCI)
M01 RR0034 (NCRR)
SOURCE: HUMAN MOLECULAR GENETICS, (1995 Dec) 4 (12) 2313-8.
Journal code: BRC. ISSN: 0964-6906.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199609
AB The ret/PTC oncogene, rearranged form of the ret proto-oncogene (**c-ret**), has been detected specifically in a minority of papillary thyroid carcinomas. Three forms of the ret/PTC oncogene have been identified; the two most common forms, ret/PTC-1 and ret/PTC-3, both result from a paracentric inversion, of the long arm of chromosome 10. In this study, we have successfully amplified the **chimeric** introns resulting from these inversions, ranging from 1.4 to 10 kb, from four of five tumors known to contain the ret/PTC-1 oncogene (where **c-ret** rearranges with the H4 gene), and from 1/1 tumors containing the ret/PTC-3 oncogene (where **c-ret** rearranges with the ele1 gene). We localized the breakpoints within the **chimeric** introns using nested PCR, and determined the exact nucleotide sequence at the breakpoint for each tumor. Our results indicate that the breakpoints in **c-ret** occur at sites distributed across intron 11, where breaks in H4 intron 1 appear more frequently at the 5'- end of the intron. Interestingly, in all tumors that we investigated, the breakpoints occurred at sits of two or three nucleotide matches between the contributing germline sequences. In summary, we describe a simple, convenient way to investigate the ret/PTC breakpoints, and have revealed several common features of the breakpoints which warrant further investigations.

L5 ANSWER 9 OF 12 Full-text? CAPLUS COPYRIGHT 1999 ACS

ACCESSION NUMBER: 1995:548539 CAPLUS
DOCUMENT NUMBER: 123:6885
TITLE: Loss of function effect of RET mutations causing Hirschsprung disease
AUTHOR(S): Pasini, Barbara; Borrello, Maria Grazia; Greco, Angela; Bongarzone, Italia; Luo, Yin; Mondellini, Piera; Alberti, Luisella; Miranda, Claudia; Aright, Elena; et al
CORPORATE SOURCE: Lab. Genetica Mol., Istituto Giannina Gaslini, Genoa, 16147, Italy
SOURCE: Nat. Genet. (1995), 10(1), 35-40
CODEN: NGENEC; ISSN: 1061-4036
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We have introduced three Hirschsprung (HSCR) mutations localized in the tyrosine kinase domain of RET into the RET/PTC2 **chimeric** oncogene which is capable of transforming NIH3T3 mouse fibroblasts and of differentiating pC12 at pheochromocytoma cells. The three HSCR mutations abolished the biol. activity of RET/PTC2 both cell types and significantly decreased its tyrosine phosphorylation. By contrast, a rare polymorphism in exon 18 does not alter the transforming capability of RET/PTC2 or its tyrosine phosphorylation. These data suggest a loss of function effect of HSCR mutations which might act through a dominant neg. mechanism. This model system is therefore capable of discriminating between causative HSCR mutations and rare polymorphisms in the tyrosine kinase domain of RET.

L5 ANSWER 10 OF 12 Full-text? MEDLINE
ACCESSION NUMBER: 94159105 MEDLINE
DOCUMENT NUMBER: 94159105
TITLE: Defects in the kidney and enteric nervous system of mice lacking the tyrosine kinase receptor Ret [see comments].
COMMENT: Comment in: Nature 1994 Jan 27;367(6461):319-20
AUTHOR: Schuchardt A; D'Agati V; Larsson-Blomberg L; Costantini F; Pachnis V
CORPORATE SOURCE: Department of Genetics, College of Physicians and Surgeons, Columbia University, New York, New York 10032.
SOURCE: NATURE, (1994 Jan 27) 367 (6461) 380-3.
Journal code: NSC. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199406
AB Receptor tyrosine kinases (RTKs) are cell-surface molecules that transduce signals for cell growth and differentiation. The RTK encoded by the **c-ret** proto-oncogene is rearranged and constitutively activated in a large proportion of thyroid papillary carcinomas, and germ-line point mutations in **c-ret** seem to be responsible for the dominantly inherited cancer syndromes multiple endocrine neoplasia (MEN) types 2A and B. The gene is expressed in the developing central and peripheral nervous systems (sensory, autonomic and enteric ganglia) and the excretory system (Wolffian duct and ureteric bud epithelium) of mice, indicating that it may play a role in normal development. Here we show that mice homozygous for a targeted mutation in **c-ret** develop to term, but die soon after birth, showing renal agenesis or severe dysgenesis, and lacking enteric neurons throughout the digestive tract. Ret is thus an essential component of a signalling pathway required for renal organogenesis and enteric neurogenesis.

L5 ANSWER 11 OF 12 Full-text? CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1995:43094 CAPLUS
DOCUMENT NUMBER: 122:102616
TITLE: The ret/PTC oncogene in papillary thyroid carcinoma

AUTHOR(S): Jhiang, Sissy M.; Mazzaferri, Ernest L.
CORPORATE SOURCE: Dep. Int. Med., Ohio State Univ., Columbus, OH, USA
SOURCE: J. Lab. Clin. Med. (1994), 123(3), 331-7
CODEN: JLCMAK; ISSN: 0022-2143
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 14 refs. The structure, function, and expression of **c-ret**, activated forms of ret oncogenes that have been identified, the transforming activity of ret/PTC oncogene, the activation of ret/PTC oncogene in papillary thyroid cancer, detection of ret rearrangement and ret/PTC1 **chimeric** transcriptors, and the clin. applications of ret/PTC oncogene in thyroid cancer are discussed.

L5 ANSWER 12 OF 12 Full-text? CAPLUS COPYRIGHT 1999 ACS
ACCESSION NUMBER: 1995:11226 CAPLUS
DOCUMENT NUMBER: 122:29119
TITLE: A t(10;17) translocation creates the RET/PTC2 **chimeric** transforming sequence in papillary thyroid carcinoma
AUTHOR(S): Sozzi, Gabriella; Bongarzone, Italia; Miozzo, Monica; Borrello, Maria Grazia; Butti, Marta Giaele; Pilotti, Silvana; Porta, Giuseppe Della; Pierotti, Marco A.
CORPORATE SOURCE: Div. Exp. Oncol., Ist. Naz. Tumori, Milan, Italy
SOURCE: Genes, Chromosomes Cancer (1994), 9(4), 244-50
CODEN: GCCAES; ISSN: 1045-2257
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Activation of the RET protooncogene tyrosine kinase (tk) by **fusion** with other genes is a frequent finding in papillary thyroid carcinoma. The tk domain of proto-RET can be fused either with the D10SI70 gene generating the RET/PTC1 transforming sequence or with sequences belonging to the gene encoding the regulatory subunit RIA of c-AMP-dependent protein kinase A, thus forming the RET/PTC2 oncogene. The authors have previously shown that an inversion of chromosome 10, inv(10)(q11.2q21), is responsible for the generation of the RET/PTC1. Here the authors report that a chromosomal translocation, t(10;17)(q11.2;q23), juxtaposes the tk domain of the RET protooncogene, which resides on chromosome 10, to a 5' portion of the RIA gene on chromosome 17, leading to the formation of the **chimeric** transforming gene RET/PTC2. The finding of the transforming protein in primary tumor cell exts. supports the conclusion that RET/PTC2 activation plays a role in papillary thyroid tumorigenesis.

=> **d l4 obib abs**

L4 ANSWER 1 OF 1 Full-text? CAPLUS COPYRIGHT 1999 ACS
AN 1998:685118 CAPLUS
DN 129:310905
TI Study and treatment of diseases related to specific cellular functions of receptor protein tyrosine kinases
IN Clary, Douglas
PA Sugen, Inc., USA
SO PCT Int. Appl., 81 pp.
CODEN: PIXXD2
PI WO 9845708 A1 19981015
DS W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG
AI WO 1998-US6842 19980407
PRAI US 1997-43207 19970408

US 1997-51715 19970703
DT Patent
LA English
AB The invention relates to methods of evaluating the specific function of a receptor protein tyrosine kinase in cells by activating the receptor in a ligand-independent fashion. In addn., the invention includes methods of identifying compds. that modulate receptor protein tyrosine kinase function. The invention also relates to a method of preventing or treating an abnormal condition caused by an aberration in the function of the **C-RET** receptor, and specifically to the treatment and prevention of neurodegenerative disorders by administering a compd. that modulates the function of the **C-RET** receptor.

=> **d his**

(FILE 'HOME' ENTERED AT 15:22:35 ON 06 OCT 1999)

FILE 'MEDLINE, JAPIO, BIOSIS, SCISEARCH, CAPLUS, EMBASE' ENTERED AT 15:27:06 ON 06 OCT 1999

L1	497	S C-RET OR C RET
L2	0	S ORPHAN C-RET
L3	32	S L1 AND (CHIMER? OR FUSION)
L4	1	S L3 AND ANTIBOD?
L5	12	DUP REM L3 (20 DUPLICATES REMOVED)